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Ascorbic acid modulation by ABI4transcriptional repression ofVTC2in thesalt tolerance ofArabidopsis

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摘要

Background

Abscisic acid (ABA) plays an important role in plant abiotic stress responses, and ABA INSENSITIVE 4 (ABI4) is a pivotal transcription factor in the ABA signaling pathway. In Arabidopsis, ABI4 negatively regulates salt tolerance; however, the mechanism through which ABI4 regulates plant salt tolerance is poorly understood. Our previous study showed that ABI4 directly binds to the promoter of the VITAMIN C DEFECTIVE 2 (VTC2) gene, inhibiting the transcription of VTC2 and ascorbic acid (AsA) biosynthesis.

Results

In the present study, we found that treatment with exogenous AsA could alleviate salt stress sensitivity of ABI4-overexpressing transgenic plants. The decreased AsA content and increased reactive oxygen species (ROS) levels in ABI4-overexpressing seedlings under salt treatment indicated that AsA-promoted ROS scavenging was related to ABI4-mediated salt tolerance. Gene expression analysis showed that ABI4 was induced at the early stage of salt stress, giving rise to reduced VTC2 expression. Accordingly, the abundance of the VTC2 protein decreased under the same salt stress conditions, and was absent in the ABI4 loss-of-function mutants, suggesting that the transcriptional inhibition of ABI4 on VTC2 resulted in the attenuation of VTC2 function. In addition, other encoding genes in the AsA biosynthesis and recycling pathways showed different responses to salt stress, demonstrating that AsA homeostasis is complicated under salinity stress.

Conclusions

This study elucidates the negative modulation of ABI4 in salt stress tolerance through the regulation of AsA biosynthesis and ROS accumulation in plants.

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